

significant. It is true that significant differences in the developmental quotient were found when various subgroups of infants were studied, such as those receiving more or less than half of their intake as maternal milk; but there remains the paradox that the mean developmental quotient of those infants who received banked human milk with a lesser contribution of maternal milk was lower than that of those fed banked human milk alone. Perhaps, therefore, the message about banked human milk and neurodevelopmental attainment is more complex than many acknowledge.

I agree that the value of banked human milk in neonatal care warrants re-examination; the pendulum has swung too far in favour of low birthweight formulas. Comparative studies examining the relative incidence of infection¹ and necrotising enterocolitis² in babies fed human milk or artificial formula suggest that differences in mortality may be apparent between the two feeding regimens. Unfortunately, the haphazard organisation of milk banks in the United Kingdom continues to prove an obstacle both to the national study of this issue and to the sophistication of processing techniques capable of controlling or changing the nutritional composition of human milk.³

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Self help organisation's advice on myalgic encephalomyelitis

EDITOR,—I should like to assure Simon Wessely¹ that neither the ME Association nor ME Action regards Michael Sharpe and colleagues' findings in patients with chronic fatigue² as another attack on its credibility. Nor do we see why the paper should "further sour relations between the organisations and the profession."

As Wessely points out, the apparent relation between functional impairment and membership of a self help organisation at follow up does not mean that membership of such an organisation is responsible for the impairment. Aside from the fact that the study did not focus on myalgic encephalomyelitis, or on the work the ME Association does, Sharpe reassured us that there was no evidence of a causal relation between membership of a patient group and level of disability.

As regards the potential damage resulting from inaccurate information about myalgic encephalomyelitis, it is worth emphasising that the quote that illustrates this came from *Nursing Standard* and not from a magazine for patients. The British organisations have long been unhappy with the way the media have portrayed the illness and reviewed existing research, and it is often extremely difficult to get erroneous or biased information corrected. Sometimes we get a right of reply, but usually we don't.

Finally, I wish to make clear that our current advice on exercise and stress is based on sound

scientific research, the recommendations of our medical advisers, and 50 years' experience. Since our aim is to help patients it would be ridiculous for us to ignore good research and to stick instead to outdated explanations, speculation, or even prejudice. No one gains from such a narrow minded approach, least of all us.

The main reason why our beliefs tend to differ from those of Wessely and H Cope and A S David³ is that the authors do not distinguish between myalgic encephalomyelitis and chronic fatigue and we do. We see myalgic encephalomyelitis as more than "mental and physical fatigue," and we have evidence that treatments that seem to help patients with chronic fatigue do not always benefit people with myalgic encephalomyelitis (C Hickie, conference "Unravelling the mystery," North Carolina, 17-18 November 1990).

Our cautious attitude may worry those who disagree with us, but a less critical approach may lead to mistakes and we are anxious to avoid this.

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Using cytokines

EDITOR,—Salem Malik and Jonathan Waxman point out that the recent increase in understanding of cytokine biology offers great promise for clinical medicine.¹ Their editorial focuses particularly on the use of cytokines as antitumour agents and on the role of these substances in the pathology of cancer. The relevance of cytokines extends to other aspects of clinical medicine in which the immune system has a role. Cytokines play an essential part in the functioning of this system, yet paradoxically they can damage tissue and be life threatening if produced in excessive amounts or under inappropriate circumstances. For example, tumour necrosis factor and interleukin 1 cause morbidity and death in conditions related to infection (for example, sepsis, adult respiratory distress syndrome, and cerebral malaria) and in chronic inflammatory diseases such as rheumatoid arthritis and ulcerative colitis.^{2,4} Advances in clinical medicine related to cytokine biology should therefore be targeted at enhancing their beneficial properties while suppressing their harmful effects. Thus, for example, in treating cancer the aim should be to retain the tumour killing properties of cytokines while suppressing their anorectic and tissue wasting actions.

These aims can be achieved by using drugs, cytokine receptor antagonists, and nutrients. The widespread metabolic changes that result from the induction or application of cytokines depend on secondary messengers and intracellular signalling. These offer broad scope for nutritional modulation.⁵ Fish oil reduces production of interleukin 1 and tumour necrosis factor in patients with rheumatoid disease and brings about an amelioration of symptoms. It is also beneficial in ulcerative colitis and psoriasis. Fish oil and saturated fats reduce the anorectic effects of interleukin 1 and tumour necrosis factor in experimental animals.⁶ Thus consideration of the nature of fat in patients' diets offers scope for manipulating cytokine biology. Recent studies showing that free radicals enhance production of cytokines draw attention to the importance of the effectiveness of the antioxidant defences of patients in whom cytokines are operating.^{6,7} Key nutrients, such as vitamins E and C, play a major part in these defences. This highlights their importance in the diets of patients

who are undergoing cytokine treatment, or are producing cytokines in amounts that are incompatible with the restoration of normal tissue function and health.

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Perineal tears

EDITOR,—Though M Stokes and D J Jones are correct to state that a perineal tear may lead to injury to the anal sphincter mechanism, they seem to imply that all tears lead to such injury.¹ They also suggest that a "prophylactic episiotomy should be performed if the perineum seems likely to tear."

Only a small proportion of perineal tears result in damage to the sphincter. A prophylactic episiotomy gives no guarantee of protecting the sphincter. A properly sutured tear is generally associated with less short term and long term morbidity than a repaired episiotomy. "Episiotomy should be used only to relieve fetal or maternal distress, or to achieve adequate progress when it is the perineum that is responsible for the lack of progress."² Advice concerning the use of episiotomy is more properly the province of midwives, other professionals concerned with intrapartum care, and the women themselves.

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Cardiac rehabilitation programmes

EDITOR,—Hannah McGee and John H Horgan note the increasing relevance of smaller uptake rates of cardiac rehabilitation programmes in older women and asks what factors might be responsible for this.¹

A short inpatient cardiac rehabilitation programme began in this district in July 1989 and was extended to include a 12 session outpatient programme in July 1990. Both of these were discontinued in April 1992 owing to insufficient funding. During the 34 months 784 myocardial events were recorded (myocardial infarctions, coronary artery bypass graft surgery, angioplasties, and heart transplants), of which 554 occurred in males and 230 in females.

A total of 62% of male patients and 63% of female patients participated in the inpatient cardiac rehabilitation programme. Later, entry to the outpatient programme was determined by a